POCKET HANDBOOK

... for the Recognition and Diagnosis of Certain Animal Diseases Exotic to Most of the Americas

The Plum Island Animal Disease Center

ARS, USDA, Greenport, N. Y. 11944

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ANIMAL DISEASES EXOTIC TO MOST OF THE AMERICAS

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ANIMAL DISEASES EXOTIC TO MOST OF THE AMERICAS

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*PRESENT IN NORTH AMERICA

**PRESENT IN SOUTH AMERICA

***NOT ILLUSTRATED

PREFACE

This booklet is a "miniaturized" summary describing the key features of and diagnostic approaches to selected exotic contagious diseases which are hazardous to livestock and poultry in the Americas. It has been kept pocket size for handy reference.

The information has been "scaled down" to serve as a memory "jogger" - first aid - for consultation incident to an emergency call to investigate a suspected exotic animal disease. Major clinical features of the disease are included. In the appended color microfiche are photographs of typical and pathognomonic lesions. The microfiche may be read in a commercial reader, by means of any 10X magnifier, under a photographic enlarger, or a dissecting microscope. Copies of the material may be made with a photographic enlarger. To obtain the most benefit from the booklet, the reader should be somewhat familiar with large animal and poultry disease syndromes generally. It is expected that the reader will wish to augment this initial short review with study of appropriate literature in depth. Examples of such literature on each disease are listed at the end of each textual description.

A more comprehensive book that covers all of these diseases and others is the "gray book" entitled "Foreign Animal Diseases," published by the U.S. Animal Health Association, 1964. (This publication is currently being revised; a new edition is expected in 1975.)

1. RINDERPEST

1. Definition:

Rinderpest (RP) or cattle plague is an acute, highly contagious virus disease, primarily of cattle, secondarily of sheep, goats, and wild ruminants. Pigs of European and North American origin, when exposed to rinderpest, may develop an inapparent infection with a mild transient fever (although they may transmit virulent virus to cattle). The American javelina and indigenous swine of the Far East are highly susceptible.

2. Etiology:

Rinderpest virus strains are immunologically uniform, but they may vary in virulence. The virus is about 300 nm in size and immunologically related to measles and distemper viruses. It is destroyed by strong acids and alkalies.

3. Geoggaphical Distribution:

The disease is enzootic in Asia and Africa but not in Europe or the Americas. The last epizootic in Europe was in Belgium in 1920.

4. Transmission:

Transmission is by contact with infected animals or indirectly with their secretions, excretions, and fomites. The virus appears in the blood and secretions before the appearance of signs. For this reason, the infection may be easily introduced

inadvertently to slaughterhouse and stockyards. Animals that recover develop solid immunity and a high antibody titer; they are not known to be carriers.

5. Hosts:

Hosts are chiefly cattle, buffaloes, deer, camels, sheep, goats, and occasionally swine.

6. Clinical Signs:

The incubation period is ordinarily 3 to 10 days, although where the disease is enzootic it may be longer; the incubation period of the experimental disease may be as short as 40 hours.

The major clinical signs are high fever, nasal discharge, erosions of the buccal mucous membranes, constipation followed by diarrhea, dehydration, rough and soiled hair coats, and death in 7 to 12 days.

7. Gross Lesions:

Lesions include punched-out-like erosions on the inner surfaces of the lower lip, the gums, ventral surface of the tongue and the soft palate. Upon necropsy, the lymph nodes are edematous. Peyer's patches are acutely inflamed, eroded, severely hemorrhagic, and necrotic. The mucosa of the abomasum is hemorrhagic. There is often edema, hemorrhage, and erosions of the mucosa of the cecum, the cecocolic junction and the rectum The mucosal surface of the last portion of the large intestine usually shows zebra stripe markings.

8. Diagnosis:

The history, signs and lesions are valuable in reaching a diagnosis. However, because of the similarity of these features to those of other diseases, discussed later, a confirmatory laboratory diagnosis is necessary.

9. Differential Diagnosis:

Diseases that resemble rinderpest clinically are acute mucosal diseases, bovine malignant catarrhal fever, acute coccidiosis, and foot-and-mouth disease. Inoculation of animals with these disease agents usually does not result in mortality. Since some strains of rinderpest are of low virulence, these other diseases may present difficulties in differential diagnosis.

10. Collection of Specimens for Laboratory Confirmation:

For virus isolation, heparinized blood, mesenteric lymph nodes, and spleen are collected early in the acute phase of the disease. One portion of the heparinized blood should be shipped refrigerated; other specimens should be received frozen at the laboratory. Serum should also be collected from animals which have been ill for the longest period of time during the outbreak.

For histopathology, specimens of tonsils, liver, spleen, kidney, and portions of intestines showing lesions should be collected in 15 percent neutral buffered formalin.

11. Laboratory Confirmation:

Attempts to isolate the virus in tissue culture or animals are carried out. Extracts from lymph nodes of infected animals may be used in the complement fixation (CF) or agar gel diffusion precipitation (AGDP) test as antigens against RP rabbit hyper-immune serum. Virus neutralization tests in cell cultures may be carried out with the sera of animals that were sick long enough to develop antibodies. A definitive diagnosis may be obtained by cross-protection tests using immune and susceptible cattle.

12. References:

Dardiri, A. H., Yedloutschnig, R. J., and Taylor, W. B. Clinical and Serological Response of American White-Collared Peccaries. . . . U.S. Animal Heath Association Proc. 73: 437-452. 1969.

Plowright, W. Rinderpest Virus. Virol. Monogr. 3: 25-110 (Ed. by S. Gard,, C. Hallauer, and K. F. Meyer) 1968. New York: Springer-Verlag.

2. AFRICAN HORSE SICKNESS

1. Definition:

African horse sickness (AHS) is a highly fatal, insect-borne, febrile, virus disease of Equidae, clinically dominated by an acute pulmonary edema or subacute cardiac form associated with localized areas of inflammatory edema and hemorrhage.

2. Etiology:

The disease is caused by a viscerotropic virus of which nine serologic types have been identified. Mouse-adapted strains are used for vaccines. More recently, murine virus has been propagated in cell cultures which are also useful as a source of relatively inexpensive vaccines for control of outbreaks.

3. Transmission:

The disease is transmitted by arthropods of various Culicoides species. The disease may persist through seasons devoid of insects (including overwintering). as well as in the absence of Equidae. The reservoir host is not yet established.

4. Hosts:

Horses, mules, and donkeys are the natural hosts. Ferrets, mice, and dogs have been infected experimentally; dogs may become transient virus carriers after eating large quantities of infected horsemeat and blood.

5. Clinical Signs:

When newly introduced into a susceptible equine population, the disease may appear in one of three forms: (1) a severe pulmonary disease with a 3- to 5-day incubation period; (2) a subacute or cardiac form associated with swellings of the head, neck, eyelids, cheeks, brisket, thorax, and ventral region of the abdomen (the most characteristic clinical sign is the prominent bulging of the supraorbital fossa); and (3) a subclinical form with a high temperature (104°F) for 1 to 2 days and a short period of general malaise.

6. Gross Lesions:

The most characteristic change is gelatinous edema of subcutaneous and intramuscular tissues, especially in the region of the temple, eyes, and throat. Edema of the lungs is common, as well as endocardial ecchymoses. In some cases there are large quantities of yellowish or sanguinous fluid in the plural cavity and pericardium. Congestion of the fundic portion of the stomach is common.

7. Diagnosis and Differential Diagnosis:

The characteristic seasonal occurrence, history, and clinical signs may assist in reaching a field diagnosis. Signs, such as edema of the supraorbital fossae, subcutaneous edems, edema of the lungs, excess of pleural and pericardial fluid are further evidence to suspect AHS.

Diseases that may be confused with AHS are anthrax, equine infectious anemia, and equine viral arteritis.

8. Collection of Specimens for Laboratory Confirmation:

Blood for viral isolation should be collected in ethylene-diaminetetraccetic acid (EDTA) or potassium oxylate, phenol, and glycerine (OCG) solutions (anticoagulent preservative) at or before the febrile period. Blood is also collected for serum 5 to 6 days following the peak of the temperature rise. At necropsy, a portion of the spleen is collected aseptically and may be placed in glycerol buffer. Other specimens, such as brain, heart, liver, and kidney, should be collected and refrigerated for bacterial examinations.

9. Laboratory Confirmation:

Susceptible equidae are inoculated intravenously with blood and spleen suspensions; these animals are observed for signs and lesions characteristic of AHS and to obtain virus and sera. The virus may be isolated by intracerebral inoculation of 2- to 6-day-old mice with diluted blood or spleen suspensions. These mice may show nervous signs in 6 to 7 days postinoculation (DPI). However, it is usually necessary to make 2 to 3 passages in mice to obtain high virus concentration in brain tissue.

The brains are harvested from mice in extremis for preparation of complement fixing (CF) antigen, which is used with reference antiserum. The test is group specific with AHS virus types. The

CF antibody is at its peak in the sera of infected Equidae 5 to 6 days after the cessation of the febrile period; following this it declines rapidly. The identification of the serotypes of the virus is done by cross-neutralization tests in suckling mice or cell cultures. It is necessary to have available for these tests all nine types of virus and their homologous antisera.

10. Reference:

Howell, P.G. African Horse Sickness. In Emerging Diseases of Animals, FAO Agr. Studies, 61: 71:108. 1963. Rome.

3. AFRICAN SWINE FEVER

1. Definition:

African swine fever (ASF) is a highly contagious, usually acute, viral disease of domestic swine characterized by fever, marked cyanosis of skin areas, and pronounced hemorrhages of the internal organs, particularly the lymph nodes, kidney and gastro-intestinal mucosa. Mortality frequently approaches 100 percent in initial epizootics.

2. Etiology:

The causative agent of ASF is a DNA virus which is 175 to 215 nm in diameter. It is sensitive to lipid solvents and Ortho-phenylphenol disinfectants but is resistant to strong acids and alkalies. The virus causes hemadsorption of swine red cells in infected leukocyte cultures. Inclusion bodies are found within the cytoplasm of cells infected with the virus.

It remains viable at refrigeration temperatures for 18 months. In 1957 the disease appeared in Portugal, presumably imported by accident from Africa. From there the disease spread to Spain. By 1967 it was reported in Italy; and in 1971 it was found in Cuba, posing a serious threat to the swine population in the Western Hemisphere.

3. Transmission:

Infection is most common as a result of contact with

recovered or carrier pigs and ingestion of contaminated or infected garbage, urine, feces, and carcasses. Recently, transmission was achieved in Africa and Spain with infected ticks.

4. Hosts:

Pigs, wart hogs, forest hogs, and bush pigs are proved reservoirs. The American javelina is resistant.

5. Clinical Signs:

In acute and subacute forms, the incubation period is 5 to 15 days. Fever, depression, lachrymal discharge, cough, diarrhea, dehydration, and death are typical signs; the course of the acute disease is 6 to 12 days.

6. Gross Lesions:

Lesions closely resemble those of hog cholera (HC), except that they may be more severe. Hemorrhages are found on the epicardium and endocardium. Lymph nodes are hemorrhagic. Spleen enlargement and splenic infarcts are common. Petechnial hemorrhages of the kidneys and urinary bladder occasionally are found.

As ASF became enzootic in Spain and Portugal, the signs and lesions in a large proportion of animals lessened in severity and were more similar to those of HC than they had been previously.

7. Diagnosis:

Disease signs and lesions may or may not be suggestive of ASF. Marked severity of lesions, especially in pigs which were

previously vaccinated for HC, may lead to a presumptive diagnosis.

8. Differential Diagnosis:

The disease should be differentiated from HC, erysipelas, and salmonellosis. Appropriate specimens must be taken for all suspect diseases.

9. Collection of Specimens for Laboratory Confirmation:

For virus isolation, spleen, gastrohepatic, and mesenteric lymph nodes are the organs of choice since they contain high virus concentrations. They are shipped to the laboratory preferably on dry ice. Where chronic ASF is suspected, serum should be obtained from swine infected longest and shipped frozen.

10. Laboratory Diagnosis:

African Swine Fever can be diagnosed in the laboratory by (1) inoculation of suspect material into immune and susceptible pigs; (2) demonstration of the hemadsorption reaction in pig buffy coat cultures inoculated with blood or spleen suspensions for the suspect pig; and (3) paired sera from the suspect animals which lived the longest may be tested by the AGDP or the immunelectroosmophoresis (IEOP) tests. However, these serologic tests necessitate the use of specially prepared cell culture antigens and known ASF immune serum. The fluorescent antibody (FA) technique for testing liver, spleen, and other frozen tissue sections and smears is also a reliable method for detection of ASF.

Recent comparative research on ASF and HC by the Commission of the European Communities (Rabot, 1971) reports the following: "Non-purulent panencephalitis...involving both the grey and the white matter is a very important characteristic of HC." This was found in 72 percent and satelitosis in 60 to 100 percent of the HC cases investigated. On the other hand, brain damage in ASF was characterized by cell degeneration, ranging from acute swelling and simple retraction to the severe cellular disease of Nissl; perivascular mononuclear infiltration was generally "not very severe".

11. References:

DeTray, D. E. African Swine Fever. Adv. Vet. Sci. 8: 299-313. 1963.

Hess, W. R. African Swine Fever Virus. Virology Monog. 9: 1-33. 1971. Springer-Verlag.

Rabot, L. B. Properties of the Virus of Classical Swine Fever. European Community, Off. Office. Pub., Pub. 8. 1971.

4. FOWL PLAGUE

1. Definition:

Fowl Plague (FP) is an acute, highly contagious, fatal viral disease of chickens and turkeys. Other birds such as waterfowl, sparrows, pheasants are also affected.

2. Etiology:

Fowl Plague is a myxovirus that can remain viable for long periods of time in infected tissues. It causes hemagglutination of the erythrocytes of chickens.

3. Transmission:

Direct contact with aerosols from infected birds is the main method of transmission. The disease is also spread by contaminated feed and equipment.

4. Hosts:

The chief hosts are chickens and turkeys, although other avian species are susceptible.

5. Clinical Signs:

Depression, drooping of feathers and tail, loss of appetite, cyanosis, and swelling of the comb and wattles are common signs.

6. Gross Lesions:

Hemorrhages in various parts of the body are common ; these are more striking in the submucosal tissues of the proventriculus. Petechiae are found on the heart, serous intestinal surfaces, and the peritoneum. Hemorrhage in the mucous membranes lining the

gizzard is also common.

7. Diagnosis and Differential Diagnosis:

Severe mortality in susceptible chickens accompanied by the signs and lesions described previously lead to a presumptive diagnosis. The snydrome must be distinguished from virulent Newcastle disease and fowl cholera.

8. Collection of Specimens for Laboratory Confirmation:

Specimens should be collected from several birds. Trachea, spleen, lungs, liver, and blood are the tissues of choice. These should be frozen and transmitted on dry ice.

9. Laboratory Diagnosis:

Virus isolation is achieved by inoculation of 9-day chicken embryos. At death, the allantoic fluid is harvested and tested for hemagglutination of chicken erythrocytes. The isolated agent is then identified by hemagglutination inhibition and virus neutralization tests using specific hyperimmune serum.

10. References:

Easterday, B. C., and Tumova, B. Avian Influenza in "Diseases of Poultry" (M. S. Hofstad, ed.), pp. 670-700. 1972. Iowa State Univ. Press, Ames.

Stubbs, E. C. Fowl Plague in "Diseases of Poultry" (H. E. Biester and L. H. Schwartz, eds.), 5th ed., pp. 813-822. Iowa State Univ. Press, Ames.

5. CONTAGIOUS BOVINE PLEUROPNEUMONIA

1. Definition:

Contagious Bovine Pleuropneumonia (CBPP) is a specific disease of cattle caused by Mycoplasma mycoides, subspecies mycoides. It is highly infectious and occurs in acute, subacute, and chronic septicemic forms.

2. Etiology:

Mycoplasma mycoides mycoides is a pleomorphic organism that is sensitive to drying and disinfectants. The causative organisms of contagious pleuropneumonia of goats and sheep share similar cultural and antigenic features with CBPP but are species specific.

3. Transmission:

The organism is transmitted through inhalation of dried bronchial secretions from infected carrier animals.

4. Hosts:

Cattle of all ages may be infected.

5. Clinical Signs:

The incubation period is usually from 3 to 6 months long but it may be shorter; in highly susceptible cattle the natural disease has been known to develop in from 10 to 14 days. Initial signs are fever, cessation of rumination, and severe cough after exercise. Other signs are an arched back, chest

pain, distended elbows, and extended head and neck. Grunting expiration, shallow rapid breathing with fluid sounds occur, then gurgling rales, pleuritic friction, and areas of dullness on percussion follow.

6. Gross Lesions:

Typical lesions found at necropsy are:

Thickening and inflammation of the pleura with occurrence of fibrin deposits. Interlobular edema in one or both lungs. The "marbled" appearance of classical descriptions is caused by distension of the interlobular septa and is accompanied by areas of gray to red hepatization. In the chronic forms of the disease necrotic areas may be walled off by connective tissue capsules forming characteristic sequestra, which may persist for a long time.

7. Diagnosis:

Contagious bovine pleuropneumonia is suspected when the marbled appearance of lobules and the presence of a large quantity of straw colored fluid in the thoracic cavity are found at necropsy.

8. Differential Diagnosis:

The lungs of animals which die of East Coast Fever may have a similar appearance to those which have CBPP. Subacute pasteurellosis sometimes may be confused with CBPP.

9. Collection of Specimens for Laboratory Confirmation:

Samples from lung lesions, pleural fluids, lymph nodes, and lung tissue exudate are collected and frozen for isolation of the organisms. Samples from lung, spleen, brain, liver, and kidney are preserved in formalin for histopathologic examination. If possible, acute and convalescent sera are obtained.

10. Laboratory Diagnosis:

Various serologic tests are used, including CF and agglutination. Metabolic and growth inhibition tests to identify the specific mycoplasma organism have been used successfully. The FA test is also used.

11. References:

Henning, M. W. Pleuropneumonia Contagiosa Bovium, Lung Sickness of Cattle, Longsiekte. Animal Diseases of South Africa, 3rd ed., pp. 204-229. 1956. Central News Agency Ltd., Johannesburg.

Hudson, J. R. Contagious Bovine Pleuropneumonia. FAO Agr. Studies, No. 86. 1971. Rome.

6. BOVINE HERPES DERMOPATHIC DISEASE

1. Etiology:

Bovine Herpes Dermopathic (BHD) disease is caused by herpes viruses that are similar in their biological, immunological, and physicochemical characteristics. Intranuclear inclusions, multinucleated, and giant cells develop in the skin of infected animals and cell cultures.

2. Transmission:

The exact mode of transmission is not known; however, biting insects and milking methods are suspected of spreading the disease.

3. Hosts:

Cattle and buffaloes of all ages are susceptible.

4. Clinical Signs and Gross Lesions:

The incubation period is 1 to 2 weeks. A fever of several days duration precedes the formation of cutaneous nodules. The nodules are first round; later they flatten and become exudative and are covered by dry scabs. When the scabs fall off, the hairless skin is normal. Bovine mammillitis lesions are chiefly restricted to the teats and udder skin and tend to become ulcerative. A large proportion of cattle herds in enzootic areas develop neutralizing antibodies without having noticeable disease signs or lesions.

5. Differential Diagnosis:

Signs and lesions are indistinguishable from those of lumpy skin disease and skin infections caused by Dermatophilus congolensis, pox, and pseudopox viruses. Lesions in the epithelium of the oral and nasal cavities cause excessive salivation, and the signs may be confused with mucosal and vesicular diseases.

6. Collection of Specimens for Confirmatory Diagnosis:

Skin lesions may contain the virus when they are fresh and exudative. A viremia is present for approximately 4 days after appearance of skin lesions. Virus may also be obtained from vesicular fluids and from exudative teat, ear, and tail lesions. Blood samples should be taken from several affected animals in early and late disease stages to obtain paired sera. The specimens should be frozen with dry ice and sent to the laboratory.

7. Laboratory Diagnosis:

To isolate virus, fluids from lesions and skin triturates from lesions are inoculated into primary bovine kidney cell cultures. The infected tissues and cultures may be examined by the electron microscope to demonstrate herpes virus morphology. Cultures stained with hemotoxylan and eosin (H and E) permit demonstration of intranuclear inclusion bodies and syncytial

cytopathogenicity. The isolated virus is identified by using reference serum from convalescent animals to conduct virus neutralization and fluorescent antibody (FA) tests. Susceptible cattle, so determined by negative serologic tests for antibodies, can be inoculated intravenously to reproduce the disease and to obtain optimal tissue and blood samples.

8. References:

Martin, W. B., Martin, B., Lander, I. M., and Pirie, H. M. Bovine Mammillitic Virus Infection (BVM) and Lumpy Skin Disease. Vet. Rec. 86: 661-662. 1970.

Breese, S. S. Jr., and Dardiri, A. H. Electron Microscopic Characterization of a Bovine Herpes Virus from Minnesota. J. Gen. Virol. 15: 69-72. 1972.

7. DUCK PLAGUE (DUCK VIRUS ENTERITIS)

1. Definition:

Duck Plague (DP) or Duck Virus Enteritis (DVE) is a disease of domestic ducks, geese, and wild waterfowl characterized by bodily hemorrhage and pathognomonic esophageal and cloacal lesions.

2. Etiology:

The disease is caused by a herpes virus approximately 180 nm in diameter with a core of 75 nm. The virus was attenuated by passage in chicken embyros and cell cultures of the same origin. The virus produces plaques in monolayer cultures. There is evidence of variation in virulence of different isolates, but it appears that all are immunologically similar.

3. Transmission:

The virus is transmitted by contact of susceptible birds with infected ones as well as both direct and indirect exposure to contaminated material and equipment.

4. Hosts:

Only birds belonging to the family Anatidae, such as ducks, geese, and swans, are known to harbor the virus.

5. Clinical Sings:

The incubation period is 3 to 7 days, and all ages of birds are susceptible. Egg production may drop 25 to 40 percent in a flock which is affected. Ducks are unable to stand, their eyes

are congested, and the feathers around the eyes are matted. When attempting to move, ducks creep with their wings outstretched. Young ducks, 2 to 7 weeks of age, show signs of dehydration and have diarrhea. Mortality may be 25 to 100 percent. A subclinical form of the disease may prevail in some flocks.

6. Gross Lesions:

In mature and young ducks tiny hemorrhagic spots corresponding to the lymphoid areas located in the esophageal mucosa appear in longitudinal rows. Later in the disease the esophageal mucosa is covered with a yellow or gray irregular pseudomembrane. Similar lesions may appear on the cloacal mucosa. In young ducklings dark-reddish, macular bands appear in the mucosal surfaces of the samll intestines. Extravasation of blood in abdominal and chest cavities as well as petechiation of the heart are associated with the disease. Secondary bacterial infections are common. The lesions in the esophagus are considered pathognomonic.

7. Diagnosis:

When present, the lesions in the esophagus may be considered adequate evidence for a diagnosis.

8. Differential Diagnosis:

This requires consideration of the possibility of exposure of a flock to other diseases such as duck virus hepatitis, fowl

plague, velogenic Newcastle disease, and pasteurellosis.

9. Collection of Specimens and Laboratory Confirmation:

Duplicate portions of esophagus, intestines, liver, and cloacal tissues with lesions are collected; one portion frozen for virus isolation and the other in a fixative for demonstration of Type A intranuclear inclusion bodies. Liver and spleen are macerated for virus isolation; the triturates are inoculated into 9- to 11-day-old duck embryos via the chorio-allantoic membranes. An isolate that is lethal to duck embryos with extensive bodily hemorrhage is suggestive of DVE. The neutralization of such an isolate by known DVE antiserum confirms the identification. An increase in serum neutralization titer of 1.75 ($\log_{10}$) in the serums of convalescent waterfowl is indication of recent infection.

10. References:

Dardiri, A. H. Attenuation of Duck Plague Virus and Its Propagation in Cell Culture. Archiv. fur gesamte Virusforschung 27: 55-64. 1969

Leibovitz, Louis. Gross and Histopathologic Changes of Duck Plague. (Duck Virus Enteritis). Amer. J. Vet. Res. 32: 275-290. 1971.

Leibovitz, Louis. Diseases of Poultry, 6th ed. pp. 732-744. 1972. Iowa State University Press, Ames.

8. TESCHEN DISEASE OF PIGS

1. Definition:

Teschen Disease (TD) is a virus disease of swine characterized by incoordination, convulsions, spasms, and paralysis.

2. Etiology:

Strains of these viruses vary greatly in virulence. The Tyrol subtype 2 virus is highly virulent while the Talfan virus in England produces either a mild syndrome or an inapparent infection. All of the viral strains are immunologically similar. The virus is resistant to drying and infective only for pigs. It is destroyed by 3% NaOH in less than 3 hours.

3. Transmission:

Transmission is essentially by the oral route although experimental, intranasal infection is successful.

4. Hosts:

Domestic swine are the only known hosts.

5. Clinical Signs:

The major clinical signs of the virulent disease are those of encephalomyelitis. They include a thermal response (104° to 106° F), loss of appetite, hyperaesthesia, muscular rigidity, convulsions, immobility, clonic spasms, paralysis of hind legs, recumbency, and death.

6. Gross Lesions:

No gross lesions are found in the nervous system; but microscopic lesions characteristic of aseptic encephalomyelitis are found, especially in the gray matter of the central nervous system. These are concentrated chiefly in the spinal cord and cerebellum and consist of perivascular cuffing and degeneration of the neurons.

7. Diagnosis:

Diagnosis depends upon the case or outbreak history, histopathology, transmission of the disease to colostrum-deprived pigs, virus isolation, and viral neutralization tests.

8. Differential Diagnosis:

Teschen Disease should be differentiated from the nervous reactions of swine which are associated with other enteroviruses, hog cholera, swine erysipelas, prolonged periods of nervousness associated with vitamin A deficiencies, and rickets caused by pantothenic acid deficiencies.

9. Collection of Specimens and Laboratory Diagnosis:

Portions of the brain, cerebellum, medulla, and spinal cord should be collected in 15% buffered formalin for histopathologic study. Samples of the same material should be obtained aseptically for virus isolation and the viral neutralization test.

10. References:

Dardiri, A. H., Seibold, H. R., and Delay, P. D. The Response of Colostrum-Deprived, Specific Pathogen-Free Pigs to Experimental Infection with Teschen Disease Virus. Can. J. Comp. Med. Vet. Sci. 30 (3): 71-81. 1966.

Grig, A. S., Bannister, G. L., Mitchell, D., and Corner, A. H. Studies on Pathogenic Enteroviruses. II. Isolation of Virus in Tissue Culture, Brain and Feces of Clinical Cases. Can. J. Comp. Med. Vet. Sci. 25: 142-160. 1961.

9. FOOT-AND-MOUTH DISEASE

1. Definition:

Foot-and-Mouth Disease (FMD) is an acute, highly communicable disease existing almost exclusively in cloven-footed animals, domesticated and wild. The disease is characterized by the formation of vesicles and erosions in the mucosa of the mouth and external nares (especially on the snout of pigs) and the skin between and above the hooves of the feet; other areas, including mammary tissue, may be involved.

2. Etiology:

The disease is caused by a virus first isolated in 1897; it is classified with the enteroviruses as a member of the picornavirus group. It has a single-stranded ribonucleic acid core with a protein coat which appears to consist of 32 capsomeres forming a symmetrical icosahedral capsid with a diameter of about 23 nm. There are 7 immunologically and serologically distinct types of virus identified as Types O, A, and C; Southern African Territories (SAT-1, SAT-2, SAT-3), and Asia-1. Within the 7 types at least 61 subtypes have been designated by CF tests.

3. Geographical Distribution:

FMD occurs in most of the major livestock producing countries of the world, except North America, Central America, Australia, New Zealand, Japan, and Ireland. Several countries in Europe,

especially Great Britain and some of the Scandinavian countries, are generally free for periods of several years; for example, FMD has not occurred in Great Britain during the last 4 years.

Type Distribution:

Types O, A, and C occur in various parts of the world while the African types, SAT-1, SAT-2, and SAT-3, were not found outside Africa until 1962 when an epizootic due to SAT-1 occurred in the Middle East. Asia-1 has been identified from Pakistan, India, Israel, Iran, Iraq, Hong Kong, Thailand, and other Near and Far Eastern countries.

4. Transmission:

The virus is transmitted by contact with infected animals (aerosols primarily), by infected animal products, and by contaminated objects.

5. Hosts:

All cloven-hooved animals, domestic and wild, are naturally susceptible; pathogenesis for some species is reduced in certain strains. The hedgehog, muskrat, armadillo, and perhaps other wild animals besides the cloven-hooved ones are susceptible; in addition, a wide variety of laboratory animals and cell culture systems can be infected with FMD virus. Man is rarely infected but is capable of transmitting the virus.

6. Clinical Signs:

In cattle, characteristic signs are a moderate pyrexia, lassitude, anorexia, excessive salivation, smacking of the lips, and drooling; these accompany the formation, rupture, and erosion of vesicles of the mouth. When the feet are involved, lameness is seen. Reduced lactation, mastitis, and abortions are common. Mortality in young animals may be as high as 50 percent but is seldom above 5 percent in adults. Swine show many similar signs; lameness with a changed gait may be quite evident. The incubation period is from 1 to 5 days or longer.

7. Gross Lesions:

Vesicles are not pathognomonic for FMD alone, since they are also associated with vesicular stomatitis (VS); vesicular exanthema of swine (VES), and swine vesicular disease (SVD). Classical vesicular lesions may not be found; when these occur they usually rupture leaving eroded, hemorrhagic, granular mucosal surfaces of the nose and mouth, as well as the skin,epithelial tissues of the feet and other regions.

Gastrointestinal lesions may be found at necropsy, particularly of the rumen. In rare cases lesions of the perineum, vulva or scrotum are seen. Tiger heart (gray, white, or yellowish myocardial lesions) may be seen in calves. In swine and sheep, lesions on the tongue are usually smaller than those of cattle.

8. Diagnosis:

Diagnosis by clinical signs is virtually impossible.

9. Differential Diagnosis:

The inoculation of susceptible horses, swine, and cattle (brought from a region distant from the outbreak) with suspect material may be helpful in differentiating one vesicular disease from another. All three of the species are susceptible to VS; cattle and swine are susceptible to FMD; swine alone respond to SVD and VES. However, laboratory confirmation is necessary.

10. Collection of Specimens for the Laboratory:

Specimens include the following:

Esophageal-pharyngeal fluid obtained with a probang desposited in sterile tissue culture medium containing antibiotic; vesicle fluids collected with aseptic technique in a sterile vial; lesion scrapings placed in tissue culture medium containing antibiotic; paired sera from individual animals or sera from separate animals taken at early and later stages. All specimens are immediately frozen for shipment (preferably) or placed in glycerol.

11. Laboratory Confirmation:

Laboratory tests for confirmation include:

Complement fixation, the AGDP, virus neutralization, and cross-immunity tests.

12. Control:

In countries where the disease is endemic, incidence of the disease is controlled by vaccination programs. In an increasing number of countries vaccination is mandatory; in others it is voluntary. In countries that are generally free of the infection, the disease is eradicated by slaughter followed by disinfection of the premises. The animal carcasses are generally destroyed by burning or burial. While costly, this method is considered to be the most effective way to deal with an outbreak.

13. References:

Bachrach, H. L. Foot-and-Mouth Disease. Annu. Rev. Microb. 22: 201-244. 1968.

Callis, J. J., Shahan, M. S. and McKercher, P. D. Foot-and-Mouth Disease. Diseases of Swine. 3d. ed., H. W. Dunne, ed., pp. 309-336. Iowa State Univ. Press, Ames.

Cottral, G. E. Diagnosis of Bovine Vesicular Diseases. J. Amer. Vet. Med. Assoc. 161 (11): 1293-1298. 1972.

10. PESTE DES PETITS RUMINANTS

1. Definition:

Peste des Petits Ruminants (PPR) is an acute, subacute, or chronic disease of goats and sheep.

2. Etiology:

The disease is caused by a virus physically and chemically similar to that of rinderpest. PPR virus also is serologically and immunologically related to the measles and distemper, as well as to rinderpest viruses.

3. Transmission:

Transmission occurs readily between sick and healthy goats and sheep, with the latter showing signs. The virus is also transmissible to cattle that are susceptible but without development of signs or lesions.

4. Hosts:

Sheep and goats are apparently the natural hosts, with the latter more susceptible.

5. Clinical Signs:

The incubation period is from 4 to 10 days. A febrile reaction is followed by watery nasal discharge which becomes mucopurulent; encrustation of the external nares accompanies drying of the discharge. The fever is followed by the appearance of mucosal lesions and by diarrhea. Females develop labial erosions. Frequently, affected animals develop respiratory signs.

6. Gross Lesions:

Lesions are found in the lungs and alimentary tract. Extensive erosive stomatitis develops in the oral mucosa accompanied by hemorrhagic gastroenteritis. The Peyer's patches are congested and necrotic; "zebra stripe" markings are prominent. Bronchopneumonia is common.

7. Diagnosis:

A presumptive diagnosis is made when an epizootic characterized by the signs and lesions enumerated above appears in sheep or goats and is accompanied by high mortality.

8. Differential Diagnosis:

Differentiation from bovine rinderpest, bluetongue, pox, and contagious pustular dermatitis is necessary.

9. Collection of Specimens and Laboratory Confirmation:

Blood, spleen, and mesenteric lymph nodes from sick animals and those in extremis are specimens of choice for submission to the laboratory. Serums from recovered animals are necessary for detection of antibodies. Cross-protection tests with susceptible goats and cattle are carried out with PPR and RP. Virus neutralization tests may be conducted in cell cultures.

10. References:

Mornet, P., Orue, J., Gilbert, Y., Theiry, G. et Sow Mamdou. La Peste des petitis ruminants en Afrique Occidentale Francaise.

Ses rapports avec La peste bovine. Rev. Elev. Med. Vet. Pays Trop. 9: 313-342. 1956.

Rowland, A. C., and Bourdin, P. The Histological Relationship Between "Peste des Petits Ruminants" and Kata in West Africa. Rev. Elev. Med. Vet. Pays Trop. 23: 301-307. 1970.

11. LUMPY SKIN DISEASE[1/]

1. Definition:

Lumpy skin disease (LSD) in classical (Neethling) form is an acute virus disease of cattle, characterized by the eruption of variable size cutaneous nodules, edema of one or more limbs, and swelling of the superficial lymphatic glands.

2. Etiology:

The disease is caused by a pox virus (Neethling) that is related serologically to sheep and goat pox virus.

3. Transmission:

Insect transmission is considered more important than is contact transmission.

4. Hosts:

Cattle and buffaloes are the natural hosts of the virus. It has been isolated and propagated in lamb testes and lamb kidney cell cultures.

5. Clinical Signs:

The incubation period is 4 to 14 days. There is a fluctuating fever, increased salivation, and nasal discharge. Skin eruptions occur following the peak of temperature rise. Skin nodules appear in different parts of the skin. They are easily seen on the neck,

1/ Lumpy Disease, Pseudourticaris, Knopvelsiekte.

back, thighs, perineum, vulva, and around the muzzle. Lesions on the muzzle, the ventral surface of the tail and ears are yellowish in color, covered with brownish lymph exudate and surrounded by an intensely congested zone. Mild lesions heal in a few weeks.

6. Gross Lesions:

The skin nodules vary in size; they are thickened masses of skin tissue of a creamy gray color, sometimes containing caseous material. In mild cases the skin lesions are round, circular, and involve the superficial skin layers, Ulcerated lesions may be found in the mucous membrane of the mouth, nose, and larynx. Similar lesions may be found on the vulva.

7. Diagnosis and Differential Diagnosis:

The nodular eruptions of the skin and mucous membranes, swelling of the limbs, and lymphatic glands are useful in making a presumptive diagnosis.

The disease must be differentiated from the Allerton type and related bovine herpes virus infections that also cause skin lesions. Other disease conditions that may be confused with LSD are allergies, screwworm infestations, and cutaneous streptothricosis.

8. Collection of Specimens for Laboratory Confirmation:

Fresh skin lesions should be harvested, and specimens of swollen lymph glands should also be collected and preserved on dry ice. Duplicate tissue samples are also preserved in formalin

for histological examination. Both acute and convalescent sera from several animals should be obtained and frozen.

9. Laboratory Diagnosis:

Cytopathogenicity and cytoplasmic inclusion bodies in cell cultures may be found. Inhibition of both features may be accomplished by known antiserum. Electron micrographic examination of skin tissues or cell monolayers may reveal the causative agent pox virus. Fluorescent antibody and ferritin tagging techniques are useful in identification of the virus particles in infected skin specimens and cell cultures.

10. Reference:

Weiss, K. E. (1963). Lumpy Skin Disease. In Emerging Diseases of Animals, FAO Agr. Studies 61: 179. 1963. Rome.

12. EPHEMERAL FEVER

1. Definition:

Ephemeral Fever (EF) is an acute, febrile disease of cattle characterized by shivering, muscular stiffness, and, occasionally, enlargement of the peripheral lymph nodes.

2. Etiology:

The disease is caused by an insect-borne virus. Blood stored at +2° C to -2° C remains infectious for about 6 weeks, but lyophilized buffy coat fractions stored at -70° C retain infectivity for at least 3 years. The virus has been adapted to infant mice and cell culture.

3. Geographic Distribution:

The disease is enzootic or has been recognized in Africa, India, Japan, the East Indies, and Australia.

4. Transmission:

The disease agent is known to be transmitted by arthropods; sand flies are regarded as the chief vector.

5. Hosts:

Bovidae are the only known hosts.

6. Clinical Signs:

The disease usually lasts about 3 days. The animal exhibits clinical signs of fever, followed by stiffness of the musculature for 1 or 2 days to a week, followed by recovery. Abortion may

occur in cows during late pregancy, and there may be a temporary interference with lactation. Mortality is 0.5% or less; stress from hot weather and foot travel may increase morbidity and mortality.

7. Gross Lesions:

At necropsy, no visible lesions have been reported in uncomplicated cases.

8. Diagnosis:

The disease may be diagnosed easily when it occurs in epizootic form. However, sporadic cases are easily confused with other diseases.

9. Differential Diagnosis:

The disease should be differentiated from mild Rift Valley fever and other febrile diseases in their early stages.

10. Collection of Specimens for Laboratory Confirmation:

Hyperemic defibrinated blood should be sent refrigerated; early and convalescent sera may be submitted frozen. Laboratory diagnosis is made by transmission of the disease to susceptible cattle or by the CF test.

To transmit the disease, the buffy coat fraction of blood from sick animals is the material of choice.

11. Reference:

Heuschele, W. F., and Johnson, D. C. Response of Cattle to Attenuated and Virulent Virus. II. U.S. Animal Health Assoc. Proc. 73: 185-195. 1969.

13. SHEEP POX

1. Definition:

Sheep Pox (SP), also termed variola ovina and Clavelee-Pockenseuche, is a highly contagious viral disease of sheep characterized by erythematous wruptions on the skin. Early in the course of the disease, SP lesions are papular but later progress to pustular eruptions. When the lesions are generalized, they may be associated with hemorrhagic inflamation of the respiratory and gastrointestinal mucosae and high mortality.

2. Etiology:

Sheep pox strains are immunologically identical but may vary in virulence. The virus is about 200 to 250 mu by 150 to 200 mu in size. Under natural conditions sheep and goat pox viruses are host specific but their immunogenic relationship has been confirmed. The virus is also related to Neethling lumpy skin disease virus and contagious pustular dermatitis virus.

3. Geographic Distribution:

Sheep pox exists in various parts of Europe, Asia and Africa. It is enzootic in Iran, India and neighboring countries. It has been reported in Egypt, the Sudan, Ethiopia and Kenya with continuing foci of the disease in Spain, Portugal and Russia.

4. Transmission:

Transmission of SP is by contact with infected sheep, their aerosols, nasal secretions, saliva or dried scabs. The disease

is transmitted by direct contact of susceptible and sick animals and indirectly by contaminated fomites and transport vehicles. The virus of SP may remain viable in wool for 2 months and on contaminated premises for as long as 6 months.

5. Hosts:

Sheep are the natural hosts for SP virus. Other hosts have been infected experimentally. Some breeds of sheep are resistant but the Marino is highly susceptible.

6. Clinical Signs:

The initial disease signs are fever, lacrimation, salivation and nasal discharge. Approximately 2 days later eruptions develop in the sparsely wooled areas of the skin such as the groin, scrotum, area below the tail, eyelids, lips, cheeks, nostrils, udder and vulvar labia. Sheep pox lesions begin as macules with a slight edema of the surrounding skin. Later the lesions develop into papules which become pustules. (The formation of pustules may or may not be preceded by the development of vesicles). As the surfaces of the pustules dry out, thin scabs are formed. The benign form of the disease is more common in adult animals with skin lesions, particularly under the tail, a mild systemic reation and mortality of about 5 to 10%. Lambs commonly experience a more malignant form characterized by depression,

generalized and coalescent skin lesions and frequently other lesions in the buccal, digestive and respiratory mucosae. Secondary bacterial infection may elicit a second temperature rise. Mortality in the severe form may reach 80% of the affected flock.

7. Gross Lesions:

The epidermal and mucosal lesions described above may be seen in the living animal or at post mortem. At post mortem the cutaneous areas surrounding the lesions are hyperemic with edema of varying degree. All, or a combination of papules, vesicles, pustules, pocks and scabs may be found. Lesions in lamb are often coalescent. Rupture of pustules before death usually resulted in matting of the wool surrounding the pustule. In the malignant form, pox lesions may extend into the mucosa of the mouth, pharynx, larynx and vagina. Small grayish lymphoma-like or caseated nodules surrounded by pneumonic areas are often found in the lung and kidney.

3. Diagnosis:

In the field, the appearance of a progressive pox or pox-like disease in a susceptible sheep flock is suggestive of SP, especially when associated with movement of animals or introduction of new stock. Clinical diagnosis of the mild form may be difficult as the lesions may be confined to small areas and be hard to detect. Laboratory assistance is necessary.

9. Differential Diagnosis:

Formation of scab-like lesions are common to SP, eczema and scabies. Eczema is non-infectious whereas the last is a parasitic disease. In their non-complicated form, none of them are associated with a febrile reaction. The mouth lesions and the systemic reaction in SP may be confused with those of peste des petits ruminants (PPR). Lack of papule and pustule formations on the skin and presence of necrotic ulcerative stomatitis in animals infected with PPR will assist in the differential diagnosis of the two diseases. Sheep pox may also be confused with contagious ecthyma (CE); however, proliferative lesions around the mouth in the case of CE as well as the use of cross-protection test may assist one in arriving at a differential diagnosis.

10. Collection of Specimens for Laboratory Confirmation:

The following should be submitted frozen with dry ice: 1) blood from sheep taken during the febrile period, 2) lymph node and lesion material, and 3) serum obtained at the acute and convalescent stages of the disease. Portions of various skin lesions should be prepared in buffered glycerin.

11. Laboratory Confirmation:

Direct light microscopy of stained smears from fresh lesions may reveal typical inclusion bodies. The electron microscope (EM) may be used to demonstrate the morphology of the virus and by ferritin tagging, the specificity of the virus. Fluorescent

antibody (FA) tests are employed with various tissues. Virus may be isolated from the blood, lymph or various lesions, (particularly during the viremic stage) by inoculation of cell cultures, chicken embryos or susceptible sheep. Detection of specific antibodies in the serum of recovered animals can be done by virus neutralization, complement fixation, agar gel diffusion precipitin, fluorescent antibody and other tests.

12. References:

Konnerup, N. (1964) Sheep Pox. Foreign Animal Diseases, Their Prevention, Diagnosis and Control. USLSA, pages 73-76.

Sabban, M. S. (1955) Sheep Pox and Its Control in Egypt. Am. J. Vet. Res. 16:209-213.

14. MALIGNANT CATARRHAL FEVER (AFRICAN)

1. Definition:

Malignant Catarrhal Fever (MCF) of Africa, also known as snotsiekte, is an acute, generalized disease of cattle and buffaloes characterized by high fever, profuse nasal discharge, severe hyperimia, diffuse necrosis of oral and nasal mucosae, leukopenia, ophthalmia, corneal opacity and enlargement of lymph nodes. Four syndromes are recognized: the peracute, intestinal, head and eye, and mild; the natural disease is usually of the head and eye form with low morbidity and high case fatality rates.

2. Etiology:

The etiologic agent of MCF in Africa is a herpesvirus with a capsid about 100 nm and an envelope about 140-220 nm in size. The virulent virus can be isolated from any tissue of the sick animal; highest titers are found in virus from the buffy coat, lymph nodes and other tissues of the reticulo-endothelial (RE) system. It is thought that a similar agent may be the cause of MCF outside Africa since the disease in other continents resembles that seen in Africa; however, no other disease agent for MCF has been isolated and the African form has not been found elsewhere.

3. Geographical Distribution:

The disease is world-wide and occurs sporadically. A

severe epizootic in cattle occurred in Colorado during the winter of 1971-1972. A sheep associated form was reported in Europe in 1798, Switzerland in 1832, the USA in 1920 and Canada in 1924. Wildebeest associated MCF was known to South Africans in the early half of the 19th century. A viral causative agent was isolated in Kenya.

4. Transmission:

The disease is transmitted from the natural reservoirs to cattle, the alien host. Wildebeest are natural reservoirs in Africa and sheep are thought to be the reservoirs elsewhere. In both sheep and the wildebeest, transmission occurs when cattle are grazed with these animals at or following parturition. Close contact between donor and recipient are regarded as essential. Studies conducted at the Plum Island Animal Disease Center and in Africa, independently, have shown that nasal discharges of infected cattle carry the African MCF virus; this finding gives a partial explanation as to how the disease is transmitted in nature and indicates that under certain conditions cattle-to-cattle transmission may occur.

5. Hosts:

The blue wildebeest (Connochaetes taurinus) and black wildeesst (C. gnu) are two known natural hosts of African MCF. They have an inapparent infection. Cattle, in which the disease appears,

are secondary hosts. Elsewhere, it is believed that sheep and cattle are the natural and secondary hosts, respectively.

6. Clinical Signs:

The clinical picture of MCF is arbitrarily divided into four forms, the peracute, intestinal, head and eye and mild forms. There is considerable overlap in the syndrome observed, which can be quite variable and the diagnosis elusive.

(1) Peracute form: Severe inflammation of the oral and nasal mucosa and hemorrhagic gastro-enteritis are observed. The course of this form is 1-3 days.

(2) Intestinal form: This form is characterized by pyrexia, diarrhea, severe hyperemia of the oral and nasal mucosa. Nasal and ocular discharge as well as enlargement of lymph nodes are common features. The course of this form is 4-9 days.

(3) Head and eye form: This is the typical clinical syndrome of MCF. The first evidence of infection is pyrexia, often heralded 2-7 days earlier by nasal and ocular discharges. Bilateral nasal discharge begins as serous and soon becomes mucoid, mucopurulent and later purulent. Encrustation is common in late stages and causes partial or complete blockage of nostrils resulting in dyspnoea. At this stage the sick animal breathes through its mouth and usually shows drooling of saliva.

The oral mucosa exhibits intense hyperemia and diffuse superficial necrosis. Because the basal layer of the epithelium is rarely involved, the necrotic lesions are designated as erosions rather than ulcers. In the live animals, these lesions have a pink or red color due to exposure of the underlying capillary bed. They are found on the lips, gums, hard and soft palate and the mucosa of the cheeks. The sharp-pointed buccal papillae are often involved and the tips slough leaving characteristic reddened, blunted papillae. Petechiae are occasionally present. These changes cause severe pain and the animal objects to the examination of its mouth.

Changes in the eye include lacrimation that becomes purulent in late stages. Ophthalmia, prominent scleral veins and swollen eyelids are common features. Corneal opacity starts at the periphery and progresses centripetally resulting in either partial or complete blindness. Corneal opacity is usually bilateral but occasionally one eye is affected more severly than the other. Photophobia is usually associated with corneal opacity. An animal exhibiting this sign closes its eye most of the time and points its head away from the source of light.

Pyrexia is a common sign of the disease and is often biphasic. The temperature is usually high, 104-107 C, and remains high until shortly before death at which time, it is subnormal.

Increased thirst starts in early stages of the disease and continues until shortly before death. Anorexia is observed in the late stages of MCF. Constipitation is a common feature of the head and eye form but terminal diarrhea is occasionally observed.

Nervous signs are rare although shivering, incordinated movements and terminal nystagmus may be observed. Skin lesions are rare. The course of this form is usually 7-14 days.

(4) Mild forms: These are syndromes caused by experimental infection of cattle using modified viruses. They are followed by recovery,

7. Gross Lesions:

Gross lesions vary according to the form and the course of the disease. Animals that die of the peracute disease usually shown no diagnostic changes.

In cases of the intestinal or head and eye form, the carcass may be normal, dehydrated or emaciated, depending on the course of the disease. The muzzle is often heavily encrusted and if wiped reveals irregular raw surface.

The respiratory system shows minor or severe lesions. There may only be a slight serous or copious mucopurulent discharge. When the course is short, the nasal mucosa shows congestion and slight to moderate serous exudate. Later, there is a profuse

purulent discharge. The mucosa is then intensily congested and edematous. Erosions may be common. Occasionally croupous pseudomembrances form and if these are removed, raw surfaces remain. Turbinates are severely inflammed and often carry pseudomembraneous exudates. The pharyngeal and laryngeal mucosae are hyperemic, swollen and later develop multiple erosions or ulcers. These lesions are often covered in part by a greyish-yellow exudate. The tracheo-bronchial mucosa is congested and usually petechiated; ulcerations may occur. The lungs are normal in peracute cases but may be emphysematous in other cases. Broncho-pneumonia may complicate chronic cases.

The alimentary mucosa may show no significant lesions in the peracute disease. Hyperemia and diffuse superficial necrosis is a common feature in other forms of the disease. The erosive lesions often envolve the tips of buccal papillae, gingivae, both divisions of the palate and the cheeks. The tongue is often normal. The esophagus may show congestion, erosions and pseudomembranes. The rumen, reticulum and omasum, do not have lesions, apart from areas of congestion. The abomasal mucosa is usually hyperemic, edematous and may have petechiae. Hemorrhagic ulcerations are also common especially in the pyloric region. The wall of the small intestine is firm and thickened by edema.

The serosa may be petechiated. The first half of intestinal mucosa may show severe congestion with blood-tinged contents. These changes decrease gradually towards the large intestine. Peyer's patches are usually normal or may show superficial necrosis. The large intestines often show minimal changes, mainly lines of congestion along the longitudinal mucosal rugae. Contents of the large intestine are scant and may be dry and pasty or stained with blood.

Characteristic lesions may appear on the kidneys. They are not always seen but when present are typical. They are usually small (2-4 mm) foci of nonsuppurative intestinal nephritis. These foci form slight rounded projections from the capsular surfaces. They are whitish and represent infiltration of mononuclear cells. The urinary bladder is often normal or its mucosa may be congested. The liver is slightly enlarged and may have miliary white foci. The gall bladder is distended but normal. The spleen is often enlarged and the Malpighian corpuscles are prominent. The heart may have petechiae on the coronary groove; the endocardium may show white patches.

All lymph nodes are usually affected but the abdominal ones are less consistently involved that those of the periphery as well as those of the head and neck. Affected glands are many times the normal size, usually 2-5 times, but occasionally up to

10 times and are usually hemorrhagic. Some, including hemolymph nodes, are usually too small to recognize in the normal animal but become quite obvious when MC strikes.

8. Diagnosis:

A history of the disease indicating close contact between the infected animal and calving wildebeests in Africa or lambing ewes elsewhere, aids a tentative diagnosis. The long incubation period of this disease, however, often shadows the association between the natural and alien hosts of MCF. Typical clinical features help in forming a preseumptive diagnosis. These include high temperature, profuse nasal discharge, severe congestion and diffuse necrosis of oral and nasal mucosa, ophthalmia, corneal opacity and gross enlargement of peropheral lymph nodes. One of more animals in a herd may be affected.

9. Differential Diagnosis:

The clinical syndrome of MCF resembles that of other diseases especially those that cause necrosis, ulcerations and erosions of the oral mucosa of cattle. Differential diagnosis should therefore include bluetongue, bovine viral diarrhea-mucosal disease (BVD-MD), rinderpest, vesicular diseases and ingestion of causatic substances.

(1) Bluetongue: The clinical reactions of MCF resembles bluetongue especially in the diffuse necrosis of oral mucosa and

crusting of the muzzle. Lameness ,common in bluetongue ,is absent in MCF and opthalmia and corneal opacity often associated with MCF are rare in bluetongue.

Virological, serological and histopathological examinations are essential for differential diagnosis of these diseases.

(2) BVD-MD: The classic clinical syndrome of BVD-MD occurs sporadically and is characterized by fever, leukopenia, diarrhea, lacrimation, nasal discharge and erosions of the oral mucosa.

Oral lesions in this disease, unlike those of MCF, are discrete, rounded or linerar depressions. Severe hyperemia and opthalmia, common in MCF are not observed in BVD-MD. Diarrhea is also rare in MCF.

Final differential diagnosis requires virological, serological and histopathological tests.

(3) Rinderpest: Rinderpest, enzootic in Africa and parts of Asia, is exotic in this country. The clinical syndrome of rinderpest is similar to that of BVD-MD. The introduction of rinderpest virus into the highly susceptible bovine population of USA would result in high morbidity and mortality rates, rapid transmission between animals and herds and a disease generally more drastic than that of MCF. Mild strains of rinderpest virus could easily be misdiagnosed as the mild form of MCF.

(4) Vesicular diseases, eg. FMD or vesicular stomatitis are excluded on the ground that these diaeases elicit vesicles on the oral mucosae, teats, and coronary bands of cattle. These vesicles rupture quickly leaving flaps of epithelium.

10. Collection of Specimens for Laboratory Confirmation:

Specimens required for laboratory examination in the study of MCF are:

1) Blood for virus isolation and cell count. Blood should be collected in EDTA (1 mg of EDTA per 1 ml of blood) or heparin.

2) Tissues for virus isolation: spleen, lymph nodes, adrenals and thyroids. (Blood and tissues for virus isolation should be refrigerated but not frozen and should be sent to the laboratory as soon as possible).

3) Tissues for histopathological studies: thin slices of kidney, spleen, liver, adrenals and lymph nodes are fixed in 10% neutral buffered formalin (in physiological saline or PBS).

4) Paired serums are required, one collected at the onset of disease and a second during convalexcence or at death.

11. Laboratory Confirmation:

Buffy coat or cell suspensions from the tissues are inoculated onto established bovine thyroid cultures which are checked for typical CPE. Although cultures may be made from the cells of infected animals which have a high viral titer, no CPE will be

observed. Cytopathic effect may also by observed by infecting bovine adrenal, kidney and testis cells as well as thyroid cells.

Animal passage may be required for final diagnosis. Viral neutralization of CPE by specific antisera may be done.

12. References:

Jubb, K. V. E . and Kennedy, P. C. Malignant Catarrhal Fever in Pathology of Domestic Animals. Academic Press, Inc., N.Y. 2nd Ed. 1970 pages 27-34.

Plowright, W. 1968. Malignant Catarrhal Fever. JAVMA, 152:795-804.

Kahrs, R. F. 1971 Differential Diagnosis of Bovine Viral Diarrhea-Mucosal Disease. JAVMA, 159:1383-1386.

15. SWINE VESICULAR DISEASE

1. Definition

Swine vesicular disease (SVD) is a contagious viral disease of swine indistinguishable in the field from foot-and-mouth disease (FMD), vesicular stomatitis (VS) and vesicular exanthema of swine (VES). It is a relatively new disease in that its first appearance was described in 1966.

2. Etiology

The infectious agent of SVD is a porcine enterovirus in the picornaviruses. The virus is a roughly spherical particle of 150 S sedimentation rate, a density of 1.34 grams per ml in cesium chloride, and a diameter of about 280 Å. It is acid and ether stabile with single-stranded RNA, stabilized at 50 C by 1 M $MgCl_2$.

The virus of SVD (SVDV) is serologically and biologically closely related to the human enterovirus, Coxsackie B-5.

3. Geographical Distribution

In 1966 a disease indistinguishable from FMD was observed in Lombardy, Italy. Failure to confirm an initial diagnosis of FMD resulted in laboratory studies which identified it as an enterovirus. In 1970 pigs in Hong Kong were vaccinated for FMD with an inactivated virus; in April of 1971 a vesicular condition, first diagnosed as FMD, was observed in the vaccinated pigs. Further studies revealed that this was the same enterovirus previously described in Italy. In 1972 FMD was diagnosed in pigs in Staffordshire, England and slaughter of swine and cattle started. Five days later, laboratory studies showed that this was not FMD but the same enterovirus previously encountered in Italy and Hong Kong. The new disease, now termed SVD, was soon iden-

tified in France, Poland, Austria and again in Italy. In late 1973, Germany and Switzerland were added. In November, 1973, the disease was reported in Japan; by 1974 it had spread to 15 different foci. The disease appears to be unchecked in both Europe and Asia.

4. Transmission

The appearance of SVD in Great Britain and other countries of Europe and also in Japan was found to be related to the recent importation of pork products or pigs from countries known or thought to have SVD in swine. In addition to the ingestion of virus in garbage, animals within herds become infected by contact with pigs shedding SVDV in their excretions, particularly the feces. Due to the viremia in SVD, all tissues contain virus and can serve as a source of infection.

The skin of pigs has been found to be much more susceptible to infection by SVD than by FMD; it is believed that viral contamination of minor wounds and scratches is a means of transmission of SVD. Pigs carried in trucks which had previously transported SVD-infected animals were infected even though the trucks had been decontaminated. Restocking proved difficult on some British farms due to reinfection. The virus of SVD is stable under a variety of environmental conditions for many months. For example, SVDV could be isolated from the surface and gut of earthworms collected from the soil above the buried carcasses of infected pigs.

5. Hosts

Swine and man are the only known species to be naturally infected. Newborn mice are readily infected by intracerebral or intraperitoneal

inoculation of SVDV but 7-day-old mice are refractory.

Several laboratory persons who had contact with SVD-infected pigs or SVDV developed a variety of illnesses traceable to infection with SVDV but not the related Coxsackie B human enterovirus.

6. Clinical Signs

Swine vesicular disease is usually first detected by the sudden appearance of lameness in several animals in a herd. On soft ground this may be overlooked. Where the animals are on hard surfaces they may be observed to limp, stand with arched back, or refuse to move even in the presence of food. These signs have maximal expression in the larger and heavier animals. The temperature is usually elevated 2 to 4 degrees C. Lesions usually appear along the coronary bands and interdigital spaces of one or more feet. Vesicles appear and rupture, resulting in ulcerous skin lesions extending to the metacarpus and metatarsus with loosening of the sole pad. Vesicles and resulting ulcerations may also be found on the snout, epithelium of the buccal cavity, the tongue and teats.

The incubation period of SVD is from 2 to 4 days for the appearance of vesicles at the inoculation sites and from 5 to 6 days for generalization of infection with vesicle formation at secondary sites. Recovery from SVD is usually rapid with pigs returning to normal within 3 weeks. Morbidity is moderate and mortality usually low. However, in the experimental infection of a sow with newborn pigs there was both high morbidity and mortality in the pigs.

7. Gross Lesions

The gross and microscopic appearance of vesicular lesions of SVD

are essentially the same as that described for FMD. No gross lesions other than those related to vesiculation have been found.

8. Diagnosis

There are no clinical signs that will help to differentiate SVD from FMD, VES or VS. In every instance regarding the initial outbreaks it is well to remember that they were diagnosed as FMD. The absence of a vesicular disease in cattle in contact with diseased pigs might be suggestive of SVD, but it should be remembered that FMD viral strains have been isolated from pigs which had very low infectivity for cattle.

Any vesicular condition should be reported and action initiated to obtain a laboratory diagnosis.

9. Differential Diagnosis

See the chapter on FMD. Presence of a vesicular condition in cattle would tend to eliminate SVD (although there could be the possibility of multiple infections in some regions). Vesicular disease in horses might suggest VS. Differential diagnosis requires the use of laboratory tests.

10. Collection of Specimens for Laboratory Confirmation

See the chapter on FMD. Vesicular fluids are collected, if available, separately from unruptured vesicles and frozen. Vesicular lesion tissues: collect about 5 grams in phosphate buffered glycerin (volumetric measurement of 5 cc of liquid may serve as a guide). Vesicular lesion materials may also be frozen. Ten ml of whole blood for virus isolation should be collected during the febrile period and frozen. Ten ml of serum should be obtained from animals in the acute and convalescent sta-

ges of the disease. Submit frozen or refrigerated.

Fecal samples from animals with and without lesions(for virus isolation) may be submitted frozen.

11. Laboratory Confirmation

Swine vesicular disease can be differentiated from FMD, VS and VES by a variety of laboratory tests such as CF, virus neutralization, differential growth in cell cultures and measurement of physical and biochemical parameters. The CF and virus neutralization tests are the most specific, and of these, the CF test is the most rapid. Antiserum against the different strains of SVDV for use in the CF test can be prepared by immunizing guinea pigs with repeated inoculations of infected fluids harvested from cell cultures or brains taken from infected newborn mice. These sera are used in a differential diagnostic CF test which also includes antisera against different types and strains of FMD, VS and VES. The test antigen usually consists of a suspension of vesicular lesion material collected from the diseased animals.

Diagnosis be use of virus neutralization can be done with the same sera used in the CF test or with sera collected from animals which recovered from the different vesicular diseases. Portions of the suspension of vesicular lesion material are mixed with each of the different sera and these mixtures inoculated into cell cultures prepared from cells susceptible to the viral infection. Diagnosis is based upon an absence of cytopathic effect (CPE) in those cultures in which the antiserum is of the same type as the test sample. Virus identification requires about 3 hours by CF and 2 to 4 days by virus neutralization.

Other laboratory methods include inoculation of a variety of cell cultures; SVDV will grow only in swine kidney cultures, while FMDV will grow in bovine kidney as well as swine kidney cell cultures. The virions of FMDV are rapidly destroyed at a pH below 6.5, while those of SVDV remain intact; if a viral agent from vesicular material is isolated in cell culture and then treated at pH 5, examination by electron microscopy will reveal particles if the agent is SVDV but none if the agent is FMDV.

12. References

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Callis, J.J., Graves, J. H. and McKercher, P.D. (1974) Swine Vesicular Disease. Proceedings of the Foreign Animal Disease Seminar Kansas State University Nov. 20 and 21, 1974. (Obtainable from the College of Veterinary Medicine, KSU, Manhattan, Kansas)

Graves, J. H., McKercher, P. D. and Ferris, D. H. (1975) Microfiche on SVD containing color illustrations of the clinical signs and lesions of SVD with extensive accompanying text and reference materials. Obtainable from the Director, Plum Island Animal Disease Laboratory, ARS, USDA, P. O. Box 848, Greenport, N.Y. 11944. Telephone: (516) 323-2500